Comment

The incidence of neonatal septicaemia caused by group B streptococci in our hospital during 1980-6 was 9·1/10 000 births, which is three times higher than the average rate in the United Kingdom.[2] Despite this, mortality during this period was only 5% (one death in 20 cases), which is one seventh the national average of 36%[2]; we ascribe this to the early antibiotic treatment of neonates with tachypnoea.

Not all babies with tachypnoea are infected with group B streptococci. The results of one year's experience with a tachypnoea monitor showed that 29 babies were referred to a special care baby unit out of the 2789 monitored; of these 29, only four had evidence of colonisation by group B streptococci and none developed septicaemia.[4] Conversely, in this series of 20 neonates with proved streptococcal septicaemia five showed no change in respiratory rate.

Though monitoring for tachypnoea led to early detection and treatment of four neonates with septicaemia caused by group B streptococci, other bacterial infections may present with a raised respiratory rate—for example, that due to group G β haemolytic streptococci.[5]

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Neuroleptic malignant syndrome in an elderly patient

The neuroleptic malignant syndrome is an uncommon side effect of neuroleptic drugs. It is characterised by hypothalamic and autonomic dysfunction, altered mental state, extrapyramidal signs, and haematological and biochemical abnormalities.[1] Mortality is roughly 20%.[2] The syndrome most commonly occurs in middle aged people, though older sufferers are being increasingly recognised. We report a fatal case in an octogenarian.

Case history

In April 1987 an 81 year old man presented with an acute exacerbation of a chronic confusional state. He had a two year history of Alzheimer's disease and non-insulin dependent diabetes. Low back pain secondary to an osteoporotic vertebral collapse was being treated with codeine. He was dehydrated and impacted with faeces, the faecal impaction being attributed to his dehydration, relative immobility, and analgesic treatment. No other abnormality was found. Initially he was agitated and required intermittent doses of parenteral haloperidol to control episodes of aggression. His constipation was corrected and his mental state improved. After psychiatric assessment he continued to receive 15 mg oral haloperidol daily on discharge from hospital.

In May 1987 he was readmitted with a four day history of increasing confusion, anorexia, and deteriorating mobility. Fever had been noted the day before admission. On examination he was found to be withdrawn and mute with his eyes tightly shut. Axillary temperature was 38°C and later rose to 39·3°C. He was sweating and had sialorrhoea. There was a sinus tachycardia of 120 beats/min. A striking finding was generalised rigidity, affecting neck, trunk, and limbs. He had irregular limb jerking and tendon jerks were brisk. Plantar reflexes were flexor. The white cell count was 15×10^9/l with a normal differential count. Thorough investigation showed no focus of infection. Serum aspartate transaminase activity was 35 IU/l and creatine phosphokinase activity 523 IU/l. Neuroleptic malignant syndrome was diagnosed and haloperidol withdrawn.

Over the next five days his muscular rigidity diminished. His level of consciousness improved and he was well enough to swallow fluids. He continued to have an intermittent low grade fever. On the fifth day after withdrawal of haloperidol he had a sudden cardiorespiratory arrest and died. At necropsy death was attributed to coronary artery atherosclerosis, though no evidence of acute myocardial infarction was found.

Comment

The typical onset, clinical features, and biochemical abnormalities in this patient allowed us to diagnose neuroleptic malignant syndrome induced by haloperidol. The clinical features consisted of fever, stupor, sweating, and salivation with extrapyramidal signs. The differential diagnosis includes catatonic schizophrenia, heat stroke, tetanus, and other infections within the central nervous system. None of these would produce the widespread abnormalities described, and infection was excluded by findings before and after death. Death was due to cardiovascular collapse in a patient with extensive coronary artery disease. This occurred suddenly at a time when signs of neuroleptic malignant syndrome were resolving.

Neuroleptic agents are used extensively in psychogeriatric practice, including the management of behavioural problems in Alzheimer's disease. There is one previous report of the syndrome in a patient with Alzheimer's disease.[3] Neuroleptic drugs are also used for non-psychotic illnesses, and one of us has reported a case of neuroleptic malignant syndrome after the use of metoclopramide as an antiemetic.[4] It is imperative that people using these drugs are aware of their potentially serious side effects.

This is the fourth case of neuroleptic malignant syndrome reported from Britain, though it is widely held that the syndrome is underrecognised. In a review of world publications Caroff found that the ages of patients ranged from 3 to 61 years, 80% being under 40. Recently there have been reports of the syndrome in men aged 65 and 72.[3,5] This case shows that people even in their ninth decade are not immune.

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Dehiscence of the infraorbital nerve as a new cause of facial pain

Many patients with facial pain and negative clinical and radiological findings receive a diagnosis of atypical facial pain. We describe a new cause for such pain—namely, dehiscence of the infraorbital nerve canal within the roof of the maxillary sinus with irritation of the nerve by inflammation of the nose and antrum.

Case reports

Case 1—A 55 year old man presented with longstanding dull pain of the upper lip, upper teeth, and cheek. He could not shave or brush his teeth without exacerbating the symptoms, and dental extractions had not relieved them. Despite an initial diagnosis of trigeminal neuralgia treatment with carbamazepine did not result in any lasting improvement. Plain radiography of the sinuses showed no abnormalities, but hypocycloidal polytomography showed a dehiscent infraorbital nerve canal (figure). Endoscopy showed that the infraorbital nerve was completely exposed, and there was mild inflammation of the antral mucosa. The symptoms could be reproduced by pressing the nerve and abolished with topical application of an anaesthetic. Repeated irrigation of the antrum and antibiotic treatment relieved the discomfort completely.

Case 2—A 50 year old woman with a long history of nasal obstruction on the right side presented with pain in the distribution of the right infraorbital nerve. Repeated courses of antibiotics had provided only transient relief. Clinical examination showed deviation of the nasal septum to the right but no other abnormality. Although plain radiographs of the sinus appeared normal, hypocycloidal polytomography showed a dehiscent infraorbital nerve on the affected side. Submucosal dehiscence of the nerve within the maxillary sinus, which had a